

L3 ANSWER 251 OF 266 CAPLUS COPYRIGHT 1999 ACS DUPLICATE 102  
 AN 1989:93290 CAPLUS  
 DN 110:93290  
 TI Effect of **interferons** and poly(I):poly(C) on the pathogenesis of  
 the diabetogenic variant of encephalomyocarditis virus in different mouse  
 strains  
 AU Giron, David J.; Agostini, Heidi J.; Thomas, Donald C.  
 CS Coll. Sci. Math., Wright State Univ., Dayton, OH, USA  
 SO J. Interferon Res. (1988), 8(6), 745-53  
 CODEN: JIREDJ; ISSN: 0197-8357  
 DT Journal  
 LA English  
 CC 15-5 (Immunochemistry)  
 Section cross-reference(s): 1  
 AB **Interferon** (IFN) can either **prevent** or exacerbate the  
 pathogenic effects of the diabetogenic variant of encephalomyocarditis  
 (EMC-D) virus. The effect seen is dependent upon the mouse strain and  
 the  
 time of IFN administration. Studies were initiated to investigate the  
 role of the IFN system in the pathogenesis of this virus infection. Here  
 IFNs or poly(I):poly(C) were administered to several mouse strains at 24  
 h  
 before or 4 days after infection with EMC-D virus. The results of such  
 treatment ranged from complete protection of the animals from the  
 diabetogenic effects of the virus to exacerbation of the infection as  
 reflected by the virus content in selected organs. The effect was  
 dependent upon the mouse strain, the type of IFN, and the time of its  
 administration in relation to virus infection.  
 ST **interferon diabetes** encephalomyocarditis virus  
 infection pathogenesis; polyinosinate polycytidylate **diabetes**  
 encephalomyocarditis pathogenesis  
 IT Mouse  
 (**diabetes** induced in, by encephalomyocarditis virus,  
**interferon** and **interferon** inducer effect on,  
 strain-dependent)  
 IT **Diabetes** mellitus  
 (encephalomyocarditis virus-induced, pathogenesis of,  
**interferon** and **interferon** inducer effect on, factors  
 modulating)  
 IT Genetics  
 (of **interferon** and **interferon** inducer effect on  
 pathogenesis of encephalomyocarditis virus-induced **diabetes**,  
 in mouse strains)  
 IT Virus, animal  
 (encephalomyocarditis, **diabetes** induced by, pathogenesis of,  
**interferon** and **interferon** inducer effect on, factors  
 modulating)  
 IT **Interferons**  
 RL: BIOL (Biological study)  
 (.alpha./.beta., encephalomyocarditis virus-induced **diabetes**  
 pathogenesis response to, factors modulating)  
 IT 24939-03-5, Poly(I):poly(C)  
 RL: BIOL (Biological study)  
 (encephalomyocarditis virus-induced **diabetes** pathogenesis  
 response to, factors modulating)

N 108:4454 CA

I Toxicity studies of human fibroblast interferon  
beta (I). Acute and subacute toxicity studies in  
mice and rats

U Shibutani, Yasunori; Obata, Masaomi; Hamada, Yoshimasa; Shichi,  
Shigeo; Ohi, Keiichi; Kaga, Nobuhiko; Yajima, Gompachi

S Toxicol. Lab., Mochida Pharm. Co., Ltd., Gotemba, 412, Japan

O Iyakuhin Kenkyu (1987), 18(4), 571-82

CODEN: IYKEDH

T Journal

A Japanese

B In an acute toxicity study, i.v. or oral  
administration of 1 .times. 10<sup>7</sup> - 2.5 .times. 10<sup>8</sup> IU and i.m.  
administration of 1 .times. 10<sup>7</sup> - 5 .times. 10<sup>7</sup> IU of human  
interferon .beta. (MR 21)/kg caused no death,  
apparent symptoms, body wt. change or abnormal autopsy findings in  
mice and rats. I.v., i.m., and oral LD50 values of MR-21  
in mice and rats were >2.5 .times. 10<sup>8</sup>, >5 .times. 10<sup>7</sup>, and >2.5  
.times. 10<sup>8</sup> IU/kg, resp. In a subacute toxicity study,  
MR-21 administered i.v. to rats for 13 wk at 1 .times. 10<sup>7</sup> - 3  
.times. 10<sup>5</sup> IU/kg/day caused no death or any symptoms attributable  
to the administration of MR-21. The no-effect dose level of MR-21  
was estd. to be 3 .times. 10<sup>5</sup> IU/kg/day under the conditions of this  
study.

L6 ANSWER 1136 OF 1213 MEDLINE  
 AN 86300315 MEDLINE  
 DN 86300315  
 TI [Alpha **interferon** in condylomata acuminata and juvenile  
**diabetes** mellitus].  
**Interferon**-alpha bei Condylomata acuminata und juvenilem  
**Diabetes** mellitus.  
 AU Gross G; Roussaki A; Ikenberg H; Drees N  
 SO DEUTSCHE MEDIZINISCHE WOCHENSCHRIFT, (1986 Sep 5) 111 (36) 1351-5.  
 Journal code: ECL. ISSN: 0012-0472.  
 CY GERMANY, WEST: Germany, Federal Republic of  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA German  
 FS Priority Journals; Cancer Journals  
 EM 198612  
 AB Persistent condylomata acuminata in a 21-year-old patient with  
**diabetes** mellitus were treated with highly purified  
**interferon**-alpha (IFN-alpha) obtained by recombinant DNA  
 technology. Daily dose was  $1.5 \times 10^6$  IU, given subcutaneously. Already  
 during treatment the condylomata regressed. Two weeks after the end of  
 therapy, i.e. after a total dose of  $10.5 \times 10^6$  IU IFN-alpha, all  
 condylomata had completely receded. Blood glucose levels remained  
 constant  
 with concomitant insulin therapy. Toxic side-effects or antibodies to  
 IFN-alpha were not observed.  
 CT Check Tags: Case Report; Comparative Study; Human; Male  
 Adult  
 Biopsy  
 Condylomata Acuminata: MI, microbiology  
 Condylomata Acuminata: PA, pathology  
 \*Condylomata Acuminata: TH, therapy  
 Diabetes Mellitus, Insulin-Dependent: MI, microbiology  
 Diabetes Mellitus, Insulin-Dependent: PA, pathology  
 \*Diabetes Mellitus, Insulin-Dependent: TH, therapy  
 English Abstract  
 Interferon Type I: AE, adverse effects  
 \*Interferon Type I: TU, therapeutic use  
 Penile Neoplasms: MI, microbiology  
 Penile Neoplasms: PA, pathology  
 \*Penile Neoplasms: TH, therapy  
 Penis: MI, microbiology  
 Penis: PA, pathology  
 Recombinant Proteins: AE, adverse effects  
 \*Recombinant Proteins: TU, therapeutic use  
 CN 0 (**Interferon** Type I); 0 (Recombinant Proteins)

TI Antibodies to . \*\*\*alpha\*\*\* .- \*\*\*interferon\*\*\* in a patient  
with systemic lupus erythematosus.  
AU Panem S.; Check I.J.; Henriksen D.; Vilcek J.  
CS Dept. Pathol., Pritzker Sch. Med., Univ. Chicago, Chicago, IL 60637,  
United States  
SO J. IMMUNOL., (1982) 129/1 (1-3).  
CODEN: JOIMA3  
CY United States  
LA English  
AB IFN is normally not demonstrable in the serum and other body fluids  
in the absence of an inducing stimulus, such as virus infection;  
however, IFN was found at high frequency in the sera of patients  
with autoimmune diseases including systemic lupus erythematosus  
(SLE), \*\*\*rheumatoid\*\*\* \*\*\*arthritis\*\*\*, and Sjogren's  
syndrome. In this report we describe the identification of  
antibodies to IFN-.alpha. (leukocyte IFN) present at a very high  
titer in the serum of an SLE patient.

L71 ANSWER 512 OF 524 COPYRIGHT 1995 DERWENT INFORMATION LTD

AN 94-302673 [37] WPIDS

DNC C94-159283

TI Use of alpha- or \*\*\*beta\*\*\* - \*\*\*interferon\*\*\* or analogues -  
for preventing or treating an autoimmune disorder, e.g. diabetes,  
arthritis, or transplant rejection.

DC B04 D16

IN SOBEL, D O

PA (GEOU) UNIV GEORGETOWN

CYC 18

PI WO 9420122 A1 940915 (9437)\* 36 pp

RW: AT BE CH DE DK ES FR GB GR IE IT LU MC NL PT SE

W: AU CA

AU 9463549 A 940926 (9503)

ADT WO 9420122 A1 WO 94-US2154 940307; AU 9463549 A AU 94-63549 940307

FDT AU 9463549 A Based on WO 9420122

PRAI US 93-26758 930305

AB WO 9420122 A UPAB: 941223

A method of preventing or treating an autoimmune disease in a mammal  
comprises administering at least one subtype of alpha- or  
\*\*\*beta\*\*\* - \*\*\*interferon\*\*\* or a hybrid or analogue of either  
or a mixt. Also claimed are:

(1) a method treating an asymptomatic preclinical autoimmune state  
in a mammal, which comprises administering a single subtype of  
alpha- or \*\*\*beta\*\*\* - \*\*\*interferon\*\*\* or a hybrid or  
analogue of either or a mixt.; (1) a method inhibiting rejection of  
transplanted islet cells or a pancreas in a mammal having  
transplanted islet cells or pancreas, comprising administering a  
single subtype of alpha- or \*\*\*beta\*\*\* - \*\*\*interferon\*\*\* or a  
hybrid or analogue or a mixt.

USE - The method can be used for treating or preventing autoimmune  
disorders such as type I \*\*\*diabetes\*\*\* \*\*\*mellitus\*\*\*,  
\*\*\*rheumatoid\*\*\* \*\*\*arthritis\*\*\*, systemic lupus  
erythematosus, scleroderma, sjogrens syndrome, mixed connective  
tissue disease, ankylosis spondylitis, Reiter's syndrome, psoriatic  
arthritis, hypersensitivity vasculitis, ulcerative colitis,  
cirrhosis, autoimmune uveitis, myasthenia gravis, Buerger's disease,  
Kawasaki's disease, systemic necrotising vasculitis, regional  
enteritis and hypoparathyroidism.

The interferon can be administered at a dose of e.g. 1x10<sup>5</sup> units to  
75x10<sup>6</sup> units, e.g. orally.

Dwg.0/2

L71 ANSWER 513 OF 524 COPYRIGHT 1995 DERWENT INFORMATION LTD

AN 93-336896 [42] WPIDS

CR 93-336897 [42]

N 108:4454 CA

I Toxicity studies of human fibroblast interferon  
beta (I). Acute and subacute toxicity studies in  
mice and rats

U Shibutani, Yasunori; Obata, Masaomi; Hamada, Yoshimasa; Shichi,  
Shigeo; Ohi, Keiichi; Kaga, Nobuhiko; Yajima, Gompachi

S Toxicol. Lab., Mochida Pharm. Co., Ltd., Gotemba, 412, Japan

O Iyakuhin Kenkyu (1987), 18(4), 571-82

CODEN: IYKEDH

T Journal

A Japanese

B In an acute toxicity study, i.v. or oral  
administration of 1 .times. 107 - 2.5 .times. 108 IU and i.m.  
administration of 1 .times. 107 - 5 .times. 107 IU of human  
interferon .beta. (MR 21)/kg caused no death,  
apparent symptoms, body wt. change or abnormal autopsy findings in  
mice and rats. I.v., i.m., and oral LD50 values of MR-21  
in mice and rats were >2.5 .times. 108, >5 .times. 107, and >2.5  
.times. 108 IU/kg, resp. In a subacute toxicity study,  
MR-21 administered i.v. to rats for 13 wk at 1 .times. 107 - 3  
.times. 105 IU/kg/day caused no death or any symptoms attributable  
to the administration of MR-21. The no-effect dose level of MR-21  
was estd. to be 3 .times. 105 IU/kg/day under the conditions of this  
study.

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